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THE RECENT ADVANCES IN THE MANAGEMENT OF PANCREATITIS, ACUTE AND CHRONIC*

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ANATOMICAL CONSIDERATIONS IN HUMANS

THE present concepts in the management of acute hemorrhagic pancreatitis (acute pancreatic necrosis), recurring acute pancreatitis, relapsing chronic pancreatitis, and calcareous pancreatitis necessitate a consideration of the anatomy of the papilla and ampulla of Vater with the relation of the pancreatic to the common duct in humans as the pathogenesis and therapy are both attributed to this relationship.

The therapy for the different forms of pancreatitis, whether it be non-operative or operative, is based upon the pathogenesis of the disease as it relates to the intraductal pressure. Glisson,[1] in 1654, has been given credit as having first described the sphincter around the common duct, and Gage,[2] in 1879, as the first to examine this sphincter microscopically. Also Claude Bernard,[3] in 1855, was aware that the common duct and pancreatic duct frequently entered the duodenum separately.

There have been several excellent articles by Opie,[4] Baldwin,[5] Mann

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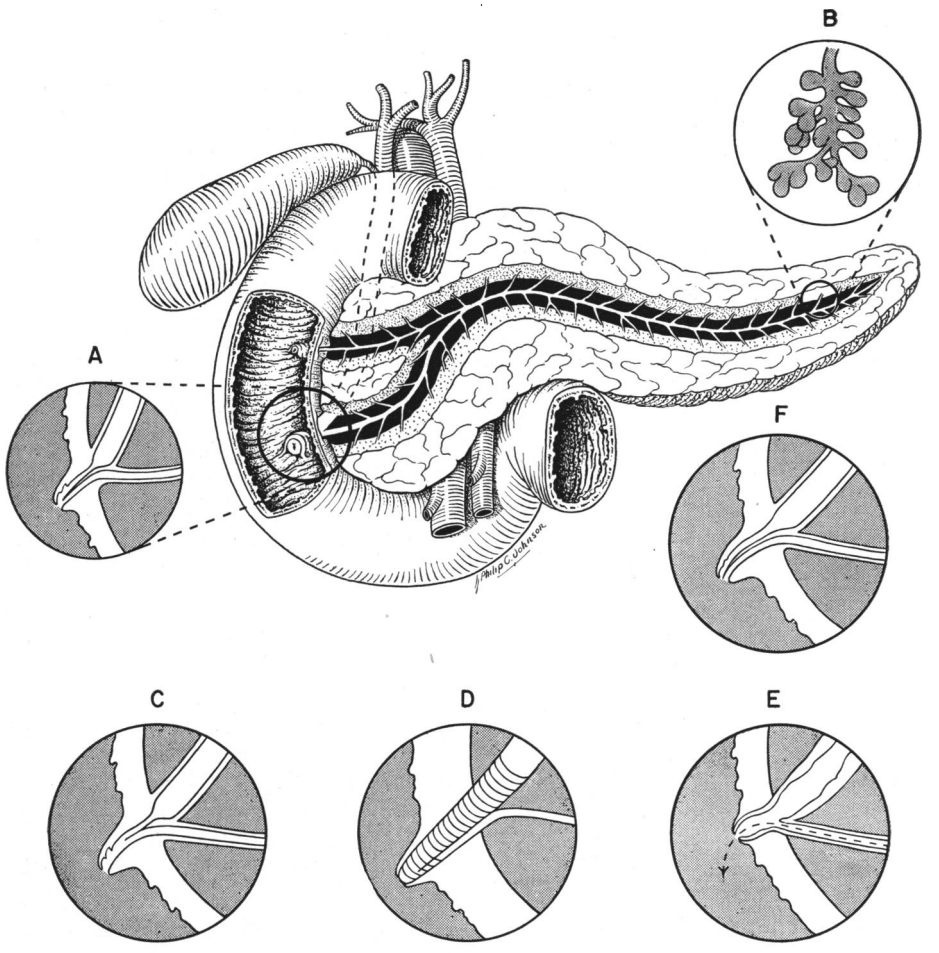

Figure 1

and Giordano,[6] and Boyden[7] describing the anatomical relationship of the common and pancreatic ducts and their relationship to the papilla and ampulla of Vater. The literature pertaining to the pathogenesis of the different forms of pancreatitis, as related to the anatomical relationship of these ducts in humans, is at variance with some of the anatomical findings, as stated by the above authors.

As recently as 1947, Boyden has emphasized that there is relative freedom from intestinal interference with the common duct and that there is a special constricting mechanism (sphincter choledochus) just above where the bile duct joins the ampulla of Vater. This has also been emphasized by Mann and Giordano[6] in their most comprehensive article

on both the human and animal studies of the relationship of the common and the pancreatic duct as related to their entrance into the duodenum (Figure 1). Opie, in 1903 in dissecting 100 humans, stated that the duct of Santorini, in 14 per cent of the cases, did not anastomose with the duct of Wirsung and in 20 per cent the duodenal end of the duct of Santorini was not patent. He found in only 30 per cent did the measurements of the pancreatic duct equal or exceed 5 mm. from the papilla of Vater which would indicate that a calculus of ½ cm. would obstruct the duct of Wirsung and would prevent a reflux of bile in 70 per cent of the human subjects. Baldwin, in 1911 dissecting 100 human adults, found that a septum divided the two ducts, and in 90 per cent, the average distance of the junction of the two ducts from the papilla of Vater was 4.8 mm. or less. In 22 per cent of his cases, there was a separate opening for the two ducts in the duodenum. Opie[8] likewise, in reporting on his cases of pancreatitis, found that in three of seventeen cases, or 18 per cent, there was a separate opening in the duodenum. Both these authors stated that the opening of the papilla into the duodenum averages 2.5 mm. Mann and Giordano, in studying 200 human subjects, found that in 31 per cent there was a separate opening for the two ducts in the duodenum, and in 4 per cent there was a fibrosis of the duct of Wirsung. In Halsted's[9] original case, reported by Opie in 1901, both emphasize that the stone to obstruct the papilla of Vater and convert the common duct and pancreatic ducts into a continuous channel must be a stone of relatively small size, namely, 3 mm., that larger stones would obstruct the ampulla, likewise obstructing the pancreatic duct.

Acute and Chronic Pancreatitis

Pathogenesis of Acute Hemorrhagic Pancreatitis: The pathogenesis of acute hemorrhagic pancreatitis (acute pancreatic necrosis) dates back to the original work of Bernard in 1855, when he injected a mixture of bile and sweet oil into the pancreatic duct, producing acute hemorrhagic pancreatitis and death within eighteen hours in the dog. Opie, likewise, injected different substances into the pancreatic duct from dilute hydrochloric acid, dilute sulfuric acid, gastric juice, and bacterial cultures and produced hemorrhagic pancreatitis, but he warned in his original experimental observations that how far this could be used to explain the pathogenesis of acute hemorrhagic pancreatitis in humans was doubtful. Likewise, the original autopsy he performed, so often quoted, revealed

the papilla of Vater occluded by a 3 mm. stone; and in this case, the pancreatic duct entered the common 10 mm. from the papilla and bile was reported in the pancreatic duct. Many have failed to realize that Opie, in trying to force bile through the papilla, put pressure on the gall bladder which may have been a post-mortem finding of bile in the pancreatic duct and not ante-mortem. Certainly, if one is to attribute bile as an etiological factor in humans, the routine procedure in autopsy examinations (namely, putting pressure upon the gall bladder) should not be resorted to.

Archibald[10] has put great emphasis on pancreatitis being due to a spasm of the sphincter of Oddi, and he performed many experiments on dogs to prove that the sphincteric mechanism was the chief offender in producing pancreatitis. He found it took a water pressure in excess of 600 mm. to overcome the spasm of the sphincter of Oddi. Likewise, the sphincter could be thrown into spasm by dilute hydrochloric acid in the duodenum and even an incision into the duodenum of dogs would throw the sphincter into spasm for as long as 30 seconds; and vagal stimulation produced a very prompt and intense contraction of the sphincter. In dogs, the maximal pressure in the common duct is 250-300 mm. of water, but after sphincterotomy, with a 70 mm. water pressure, it readily entered the duodenum and this would last for as long as an 8 week period. Other investigators, notably Deaver[11] and Braithwaite[12] have attributed pancreatitis to the spread of infection by the lymphatics from the gall bladder to the pancreas. The lymphatics take the infection to the lymph glands along the cystic duct, then by the periductal lymphatics to the nodes along the common duct and to the head of the pancreas. Likewise, the hematogenous route has been suggested as a possible cause of pancreatitis, and the pancreatitis associated with mumps has been used to substantiate this concept. In view of the fact that mumps is probably a viral infection, it would lend rather limited support to the hematogenous concept of pancreatitis. Flexner,[13] in 1906, performed experiments on dogs to prove that the crystalline principles of the bile, namely, the bile salts consisting of taurocholate and glycocholate were more injurious to the pancreas than the bile containing chiefly the colloid or mucin and he added mucin, agar, and gelatin, all of which greatly diminished the injurious effect of the bile when injected into the pancreatic duct of dogs. Nordmann [14] closed the ampulla of Vater with a purse string suture with a resulting jaundice

without the occurrence of acute hemorrhagic pancreatitis. The pancreatic duct was dilated and contained bile. When virulent bacteria were introduced into the pancreatic duct, acute hemorrhagic pancreatitis resulted. Mann and Giordano,[6] in their very comprehensive animal studies, concluded that pancreatitis will not be produced by occluding the common duct when there is a very obvious opening of the pancreatic duct into the common duct, namely in goats. They found that acute hemorrhagic pancreatitis could not be produced in animals unless the pressure in the duct was 800 mm. of bile.

Also bile could not be forced into the pancreatic duct with a pressure less than 500 mm. of bile.

If one relies solely on the intraductal pressure produced in animals which is produced by the secretory mechanism of the liver, the contractility of the gall bladder, and the resistance of the duct system to a completely occluded common duct, acute hemorrhagic pancreatitis is not produced unless the solutions are injected directly into the pancreatic duct under increased pressure. This observation has also been emphasized by Rich and Duff[15] and they state that unless the ductules and acini are ruptured by the increased pressure, the acute hemorrhagic pancreatitis is never produced. Mann and Giordano[6] conclude that the pressure in both the common duct and the pancreatic duct are essentially the same and they used a manometer with a cannula in each duct where the pressure could be measured and expressed in mm. of bile. They found that the maximal intraductal pressure in both organs is between 250 and 350 mm. of bile. Likewise, by stimulating the liver with intravenous sodium salicylate, bile did not seem to enter the pancreatic duct when the pressure was 300 mm. of bile, but by stimulating the pancreas with secretin, pancreatic juice entered the common duct under 100 mm. of bile pressure in the pancreatic duct.

Rich and Duff,[15] in studying 24 patients dying from acute hemorrhagic pancreatitis, found a calculus in the ampulla of Vater in only two of the patients coming to autopsy and two had bile in their pancreatic duct, but they state that pressure had been exerted on the gall bladder which may have explained the presence of bile in the pancreatic duct. They state they could find no experiment in which bile caused acute hemorrhagic pancreatitis without rupture of the ducts or the acini of the organs which had resulted from an abnormally increased pressure in the ductal system. They injected 1 cc. of a solution of tryp-

sin dissolved in saline made of beef pancreas subcutaneously in dogs after it had been run through a Berkefeld filter and proved to be a sterile culture and it would cause hemorrhage and edema, but did not cause fat necrosis. The vascular lesions were typical of those seen in humans in acute hemorrhagic pancreatitis. Cultures taken did not reveal any bacteria at the site. One cc. of pancreatic juice from the duct of a dog subcutaneously produced fat necrosis due to the lipase, but no vascular lesions. If the dog was starved for two days and fed a meal of meat and milk three to four hours before the experiment and then injected with 8 cc. of pancreatic juice, it produced fat necrosis and typical vascular lesions; and they conclude that a permanent fistula in the pancreatic duct may give such diluted pancreatic secretions as to have little or no necrotizing activity, but when the pancreatic juice is collected during a large meal consisting of proteins, it may have great necrotizing activity.

They stated that inactive trypsinogen may be converted into trypsin without being activated by enterokinase in the succus entericus of the duodenum and they have obtained identical lesions from pancreatic juice which has never been in contact with the duodenum and two explanations are offered. One, that in certain instances, pancreatic juice may contain active trypsin; secondly, the trypsinogen in the pancreatic juice may become activated by proteolytic activity when it escapes into the tissue. This might possibly be explained by the fact that the calcium ions in the tissue fluids may activate the trypsinogen into trypsin. They feel that metaplasia of the duct epithelium may be the explanation of acute hemorrhagic pancreatitis. The localized proliferation of the epithelium causes it to lose its cuboidal and columnar character and assume a transitional or basal epithelium. These masses may partially or completely obstruct the duct. They found in 24 autopsies of patients dying from acute hemorrhagic pancreatitis in thirteen of these patients there was evidence of metaplasia. Likewise, in 150 routine autopsies, in only twenty-eight or 18 per cent was there metaplasia of the ducts. Also in the patients dying from acute hemorrhagic pancreatitis, they found that in the thirteen with metaplasia 70 per cent of them had no evidence of gall stones or cholecystitis. Likewise, in the twenty-eight patients with pancreatitis, four or 15 per cent had cholecystitis and cholelithiasis.

Diagnosis of Acute Hemorrhagic Pancreatitis: The diagnosis of acute hemorrhagic pancreatitis is not always possible unless a laparot-

omy has been performed. The most helpful laboratory procedure is the finding of an elevated serum amylase in the early stages of the disease. This is not pathognomic of acute hemorrhagic pancreatitis, as it may occur in acute perforated ulcers. Musgrove[16] has reported on elevated serum amylase in patients with perforated gastroduodenal lesions, and the differential diagnosis of these two conditions is most difficult at times. Of course, if air is present under the diaphragm, the diagnosis of a perforated viscus is reliably established, but many patients with a perforated viscus will not have air under the diaphragm. Another diagnostic aid is an upper abdominal paracentesis which may reveal hemorrhagic fluid with marked elevation of the amylase in cases of acute hemorrhagic pancreatitis. The most fulminating forms of acute pancreatitis are more easily diagnosed, but those of lesser severity are more difficult.

Diagnosis of Chronic Pancreatitis: One is inclined to confuse the post-cholecystectomy syndrome with relapsing chronic pancreatitis. Dreiling[17] has used the secretin test as a means of determining the presence of chronic pancreatitis in patients with a post-cholecystectomy syndrome. Elman[18] has emphasized chronic pancreatitis in patients presenting this syndrome, but in ninety-eight of Dreiling's cases, averaging 5.6 years post-operative, he found in only two, an abnormal secretin response; one proved on second operation to have carcinoma of the pancreas and the other patient had a chronic pancreatitis. Gross, Comfort, Wollaeger and Power[19] found the secretin studies in patients with parenchymatous hepatic disease suggested a hypersecretion and not a hyposecretion, which gives more clinical evidence to a possible reflux of pancreatic enzymes in hepatic disorders. They also stated that steatorrhea is not due to insufficiency of external pancreatic secretion. Six of nine patients had steatorrhea with a normal external pancreatic function. Colp and Doubilet[20] called attention to the intraductal pressures in pancreatic ducts being more likely to reflux pancreatic juice into the bile duct than the reverse. In both the acute fulminating forms of pancreatitis and the chronic relapsing forms, one is unable to establish a diagnosis with certainty in a high percentage of cases.

Treatment of Acute Hemorrhagic Pancreatitis: Acute fulminating forms of hemorrhagic pancreatitis are being treated in many hospitals and clinics by conservative management and when conservative management is elected, one should add to his armamentarium a blocking of

the vagus by either intravenous banthine or atropine given hypodermically. Shingleton, Fawcett and Vetter[21] have shown that banthine will reduce the external pancreatic secretion when the pancreas is stimulated by secretin. One, in this way, can paralyze the sphincteric action, which has been demonstrated by Archibald, to raise the intraductal pressure. If the bile reflux has any relationship to the cause of pancreatitis, this would diminish its possible aggravating effect. Schaffarzick, Ferran and McCleery[22] ligated the pancreatic ducts in dogs with a marked rise in serum amylase after four days, but less marked in vagotomized dogs. Likewise, the patients should have continuous gastric suction so as to reduce to a minimum the amount of acid coming in contact with the duodenum, which in turn causes a spasm of the sphincter of Oddi with an acceleration of the disease if it is on a reflux basis and, obviously, food should be withheld for the same reason. It has been shown by Babkin[23] that glucose stimulates pancreatic secretion and if it is used intravenously, it should be covered by insulin.

Edmondson and Berne,[24] Lipp and Hubbard,[25] and Ochsner[26] have stressed the low calcium findings after the third to the tenth day in patients suffering from acute hemorrhagic pancreatitis, and Ochsner has suggested calcium replacement in the early stages of the disease to overcome the calcium deficiency. Whether this should be done or not has a certain theoretical objection, namely, that the tissue calcium may be a factor in transforming trypsinogen into trypsin in the pancreatic tissue.

Turner[27] observed that patients suffering from acute hemorrhagic pancreatitis might have severe ecchymosis in either the right or left flank or both and around the umbilicus. Some of these patients had the area incised with sterile cultures. This would suggest that the retroperitoneal escape of the pancreatic secretions to the flanks might have been activated to trypsin by the calcium ions in the soft tissues.

Splanchnic blocks have been used as a means of treating patients in the acute stages of this disease and it is quite effective in relieving them of the severe pain. If the patient can be relieved of the pain without the use of morphine or its derivatives, it might be beneficial, providing that the intraductal pressures are not unduly increased. It is well known that sphincteric pressure is increased with the usage of morphine. Curreri and Gale[28] have had a special recording apparatus constructed by Gilson in the Department of Mechanical Electronics at the University of Wis-

consin and their observations indicate the intraductal pressure is increased by splanchnic blocks. In the patient treated conservatively, the use of aureomycin seems definitely indicated. Persky, Schweinburg, Jacob and Fine[29] stated that when acute pancreatitis was produced experimentally in dogs and aureomycin used both pre-operatively and post-operatively, after ligating the pancreatic duct, the operation did not result in a fatality.

Operative Treatment: A very high percentage, if not the majority, of clinics are advocating non-operative management of the fulminating forms of acute hemorrhagic pancreatitis, but so far in these critically ill patients, there has been no appreciable change in the mortality rate. It still ranges, regardless, of whether the patient is treated by operation or by non-operative measures, between 50 and 65 per cent.

If the patient is operated upon because of a mistaken diagnosis or by choice, a decompression of the biliary system should be accomplished, the beneficial effects of which may be attributed more to preventing a reflux of the active pancreatic ferments into the biliary tract than the reverse. Walters and Marshall[30] found pancreatic ferments from a T tube of sufficient concentration to cause skin digestion in four patients. Colp, Gerber, and Doubilet[31] found pancreatic ferments in the gall bladder in three cases of acute cholecystitis. Reid[32] diverted the pancreatic juice in the gall bladder experimentally in dogs by operation, without harmful effects, but if the cystic duct was ligated, the animals developed an acute chemical cholecystitis. The most satisfactory means of adequate drainage is by means of a choledochotomy in non-calculous biliary tract disease. If stones are present in the gall bladder, the patient may have a cholecystectomy and decompression of the biliary system through the cystic duct, but if the patient is too ill for a cholecystectomy, a cholecystostomy may be performed. In recurring acute pancreatitis, the relapsing chronic pancreatitis, and calcareous pancreatitis, we have many methods of surgical attack. In Archibald's original observations, he considered a division of the sphincter of Oddi as a method of preventing these recurring episodes, but in the light of anatomical knowledge and secretory pressures in the two ducts, it makes one question this method of attack. Doubilet and Mulholland[33] are advocators of this procedure. They do not report post-operative secretin studies to prove the pancreas has regained any external secretory function. Likewise, there are many other procedures being advocated, such as vagotomy, thoracolumbar

sympathectomy, celiac ganglionectomy, subtotal gastrectomy plus vagotomy, choledochojejunostomy with Roux "Y" anastomosis, even total pancreatectomy and subtotal pancreatectomy. Which of these procedures is the operation of choice depends to a certain extent on whether one accepts the reflux theory of pancreatic disease.

A consideration of the reflux theory or common channel theory dates back to Halsted's original case, where he suggested that the mechanism was similar to the hydraulic ram principle, namely, that the common duct was the feed pipe, the pancreatic duct the delivery pipe, and the stone in the papilla, the stop cock in the mechanism. Sufficient emphasis has never been placed on the fact that the pancreas has a greater secretory volume in 24 hours than does the liver in humans. It is estimated that in the 70 kilo man his pancreatic secretion in 24 hours is perhaps 1500 cc.[34] while likewise, the bile secretion would perhaps not exceed 600 to 750 cc.[35] That being the case, it would seem that the secretions from the pancreas could enter the common duct much more readily than for the bile to enter the pancreatic duct. Mann and Giordano have proved in animals that this is true and Colp and Doubilet[20] and Doubilet and Mulholland[33] suggested the same is true in humans. Therefore, before deciding on an operative procedure which is based on the reflux or common channel theory, one should re-evaluate the reasons behind the different operative procedures.

Treatment of Chronic Pancreatitis: Most of the patients in whom the diagnosis of chronic pancreatitis is made seek relief for one symptom, namely, pain. By using the secretin test, Dreiling[17] has studied a group of patients with a post-cholecystectomy syndrome. The secretion test has shown that only a very small number of patients, namely two, have had any evidence of an abnormal pancreatic secretin test. Even if one can be 100 per cent sure that the pain is entirely due to the pancreas, one is likewise confronted with choosing a procedure which has the greatest likelihood of relieving the patient of his complaints.

Operations: Vagotomy and thoracolumbar sympathectomy were first performed by Rienhoff and Baker[36] to relieve a patient from pancreatic lithiasis and chronic pancreatitis. In their original procedure, they removed from the fifth thoracic through the second lumbar ganglion with the splanchnic nerves bilaterally plus a bilateral vagotomy.

McCleery, Kesterson and Schaffarzick[37] performed vagotomy on eleven patients with recurring acute pancreatitis, as they felt psycho-

genic stimuli played an important role in the etiology of this disease. There have been many advocates of operative procedures on the sympathetic system for the treatment of patients suffering from chronic recurring pancreatitis. Notably among those have been Mallet-Guy,[38] Mallet-Guy and de Beaujeu,[39] Smithwick,[40] Ray and Console,[41] and de Takats, Walter and Lasner.[42]

Ray and Neill[43] feel that the pain sense in the pancreas and the other upper abdominal viscera is mediated wholly by the visceral afferant nerves which accompany the sympathetic nerves and the pain is eliminated by a bilateral thoracolumbar sympathectomy. Ray and Console[41] feel that removal of the eleventh thoracic through the first lumbar bilaterally with the splanchnic nerves will completely denervate the pancreas of all pain sensation. Vagotomy plays no role in relieving the patient of the pain sensation, but it affects the external secretion of the pancreas as has been demonstrated both experimentally in animals and clinically.

Subtotal gastrectomy with sub-diaphragmatic vagotomy has been advocated by Richman, Colp, and Lester[44] to diminish the free hydrochloric acid, which in turn causes a spasm of the sphincter of Oddi. This procedure, of course, eliminates the gastric stimulation as well as the cephalic phase of gastric secretion. The external secretion of the pancreas would be diminished as a result of the elimination of the acid chyme coming in contact with the duodenum and producing a spasm of the sphincter of Oddi and also because of the vagal interruption; they report three cases treated this way.

Bowers[45] advocated choledochojejunostomy with a Roux "Y" anastomosis and by such a method, bile is shunted away from the sphincter, and he reported six cases. Whipple[46] advocated total pancreatectomy in calcareous pancreatitis. Mallet-Guy and de Beaujeu[39] feel that left-sided unilateral splanchnicectomy is a satisfactory procedure in the relief of a patient suffering from chronic pancreatitis. Grimson[47] advocated bilateral celiac ganglionectomy, and Ray and Console[41] advise a minimal bilateral thoracolumbar sympathectomy and splanchnicectomy. Rienhoff and Baker[36] advocate an extensive sympathectomy plus vagotomy, and Doubilet and Mulholland[33] sphincterotomy.

In considering the operative procedure, one should consider whether there is a possibility of increasing or diminishing the external function

of the pancreas by the operation or whether it is purely for the relief of pain without any injurious effects upon pancreatic function. It would seem, in reviewing the many procedures which have been advocated, one should consider what the end results of some of these procedures may have upon the external pancreatic secretions. The evidence so far does not indicate that dividing the sphincter of Oddi has changed the external secretion of the pancreas. It is our feeling that the procedure of choice at the present time is one which offers complete assurance that the patient can be relieved of his pain with no injurious effects resulting to the external pancreatic secretion.

With that in mind, one of our Resident Surgeons, Robert Pfeffer, has studied thirty individuals; ten normals, ten having had rather extensive thoracolumbar sympathectomies for hypertension from at least the fifth thoracic through the third lumbar with the greater, lesser, and least splanchnics also removed, and ten with transthoracic vagotomy. In the sympathectomy group, the average post-operative period at the time of study was 3.8 years, and in the vagotomy group, the average was 4.8 years at the time of study.

After secretin stimulation alone, the normal group compares favorably with the findings of Agren and Lagerlof[48] and Dreiling and Hollander[49] who also used an 80 minute collection period. The sympathectomy group has maintained function quite comparable to the normals with both groups experiencing an increased output of enzyme following vagal stimulation. The overall response in the sympathectomy group was 93.8 per cent rise as against a 98.5 per cent rise in the normals with both groups showing an increased concentration of enzyme. This is in marked contrast to a decline in enzyme activity of 337.5 per cent following vagal stimulation in the vagus resection group with a corresponding decrease in concentration.

The vagus resection group remained below the normal and sympathectomy group in all respects. In the resting state, these individuals produced less volume and less enzyme. When stimulated with secretin, the output remained sub-normal. In one of these patients, a previously unsuspected diagnosis of chronic pancreatitis was made. After stimulation with secretin and insulin, these differences became progressively more accentuated with the vagus resection group showing clear signs of pancreatic exhaustion. Secretion in the last 40 minutes of the test almost came to a standstill and enzyme values were exceedingly low. On the

basis of this test, eight out of ten patients studied had values for volume HCO_3—and enzyme output in the diagnostic range of chronic pancreatitis, with the other two patients but a little higher.

Serum amylase values at the end of each test were compared with prestimulation values to check the possibility of pancreatic edema developing with subsequent obstruction of the ducts. None of these values reached such diagnostic levels.

The results of these tests indicate that the vagus nerves have a decisive effect on the production of pancreatic enzymes in man. After an average period of four years, eight months, the vagus resection group revealed sub-normal pancreatic function, both in the resting state and following secretin administration. When placed under maximal stimulatory stress with the hormone secretin and vagal excitation, there is not only a statistically significant decrease in volume and enzyme output and concentration, but clear signs of pancreatic exhaustion.

In sharp contrast to this, the sympathectomy group has maintained pancreatic function about normal after an average post-operative period of three years, eight months. The sympathetic and splanchnic nerves seem to have little effect on the output of pancreatic juice in man. Shingleton, Fawcett and Vetter have recorded similar findings.

These basic physiological concepts have bearing on the clinical treatment of both acute and chronic relapsing pancreatitis. Blockage of the vagal impulses with anti-cholinergic drugs (i.e. atropine or banthine) as part of the conservative management of acute pancreatitis seems well warranted. That stimulation of these nerves in an acute situation does increase the output and concentration of enzymes has been demonstrated. Whether vagotomy is indicated in the treatment of the chronic forms of pancreatitis seems most questionable.

Comment: The operative treatment at this time should offer the maximum assurance of the relief from pain with the greatest assurance that the external secretion of the pancreas will be restored to normal, or at least not diminished, in subsequent years. Thoracolumbar sympathectomy with splanchnicectomy seems to offer complete relief from pain without any deleterious effects on the external secretion of the pancreas. The magnitude of the procedure depends upon whether the diagnosis of chronic relapsing pancreatitis can be definitely established without other organs in the upper abdomen not playing a role in the pain mechanism, such as the post-cholecystectomy syndrome. Therefore, if

one does a more radical thoracolumbar sympathectomy, sixth thoracic through the third lumbar with a bilateral splanchnicectomy, one is assured of relief of pain whether it be due to biliary dyskinesia or true chronic pancreatitis.

This procedure has been the one of choice in the past nine patients with chronic relapsing pancreatitis or calcareous pancreatitis with gratifying results.

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